

HEADACHE: ITS ETIOLOGY AND MANAGEMENT

*Transcription of a Panel Meeting on Therapeutics**

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MODERATOR MERRITT: The subject for discussion this afternoon is headache. We have a panel of experts to discuss various aspects of the question. Unfortunately the subject is so broad that time does not permit us to bring to you all of the experts who would be needed to cover it fully.

Headache is one of the most common symptoms to which man is subject. There are few individuals who have not suffered from headaches on several or many occasions. Headaches may occur without any organic pathology or they may be a manifestation of serious disease. In the majority of cases headaches are transient and their occurrence can be directly related to an acute febrile episode or some other definite cause. The situation is different, however, in those cases where the headaches are chronic or occur at intervals over a period of months or years. It is important in these cases to determine by thorough study the underlying cause of the headache in order to correct any serious pathology. Headaches are a common manifestation of intracranial tumors, infections, head trauma, febrile illnesses, arterial hypertension, cerebral arteriosclerosis, cerebral anoxia or asphyxia of any cause.

All of the above enumerated conditions account for symptoms in only a small number of patients who consult their physician for chronic headaches. In many of these a thorough study will not reveal any significant finding which will explain their headaches. In the majority of patients with headaches of unknown cause, the symptoms are due to migraine or related to psychological tension. The pain-sensitive structures in the head are tissues covering the cranium, the large intracranial venous sinuses and their tributaries, the dura mater at the base of the skull, the dural arteries, the large arteries at the base of the brain, the 5th, 9th and 10th cranial nerves and the upper cervical nerves.

The substance of the brain, the small arteries on the surface of the cortex and the dura over the convexity of the brain are all relatively insensitive to pain.

Stimulation of the pain structure on or above the tentorium results in pain or headache in front of a line directly drawn vertically from the ears across the top of the head. This pain is transmitted by the 5th nerve. Stimulation of pain structures below the tentorium causes pain behind the line described and is transmitted by the 9th and 10th cranial nerves, and the upper three cervical nerves.

With this brief introduction, I would present to you the members

of the panel who will discuss the aspects that are appropriate to their specialty.

Our first speaker this afternoon is Dr. Daniel C. Baker, Jr., Associate Clinical Professor of Otolaryngology, College of Physicians and Surgeons, Columbia University, and Associate Attending Otolaryngologist, Presbyterian Hospital.

DR. DANIEL C. BAKER, JR.: The nose and the sinuses are considered by the lay person to be the most common cause of headache. The otolaryngologist is frequently consulted by the patient before any other physician sees him. In taking a routine history of a patient, regardless of the nature of his illness, if the question is asked, "Do you have headaches", the patient will usually reply "Yes". If you ask the patient what he thinks the cause of the headache is, he will say he has a "sinus".

In our civilization most people have some complaints relative to their nose and throat. Nasal blockage, sneezing, postnasal discharge, clearing the throat and cough are common symptoms. Humidity, temperature changes, heat and cold, dust, chemical content of the air, allergic factors, tension, mental fatigue, excessive smoking and other things may cause nasal symptoms. These symptoms represent the reaction of the individual to his environment.

It is possible to have extensive sinus disease and nasal disease and not have the symptom of headache.

In order to understand why the sinuses are thought to be a cause of headache, a knowledge of the anatomy of the paranasal sinus system is necessary. At birth the maxillary sinuses are already present and so are the ethmoid cells. The frontal sinuses are present after the first year of life and the sphenoid sinuses hollow out of the sphenoid bone in the fourth year. In a six year old child the sphenoid sinus has already taken on a definite relationship with the cavernous sinus and its combined structures.

On the lateral wall of the nasal chamber, the inferior turbinate has large venous spaces. It is this vascular tissue that causes nasal blockage and headache, when it is congested. There is a small amount of this tissue in the middle turbinate. All of the anterior group of ethmoid sinuses drain into the middle meatus which is covered by the middle turbinate. Any blockage of drainage or any interference with ventilation can cause headache.

The sphenoid sinus has a definite relationship with the optic chiasma and the hypophysis. There are numerous dehiscences in the lateral wall of the sphenoid sinus.

The nasopharynx is a common location for carcinoma to develop. There is an anaplastic type of cancer (Schmincke's tumor) which instead of growing out into the nasopharynx will grow into the different recesses and also the foramina at the base of the skull. It is an invasive type of carcinoma which causes very severe head pain along the branches of the first, second and third divisions of the fifth nerve.

The interior of the nose is innervated by the anterior ethmoidal and the maxillary portion of the trigeminal nerve. The posterior part of the nasal cavity, including the nasal septum gets the major portion of its nerve supply from the second division of the fifth nerve by way of the sphenopalatine ganglion. These nerves pass through the sphenopalatine ganglion to their distribution. Nerves from the ganglion pass not only to the nasal cavity, but also down the descending palatine canal across the hard palate and then branch upward into the anterior part of the nose through the foramina in the premaxillary wings. The vidian nerve, which is a branch from the carotid plexus and also the facial, joins the sphenopalatine ganglion. It passes through the lateral wall or the floor of the sphenoid sinus.

The teeth can cause the symptoms of sinusitis and headache. The upper three molar teeth always have a relationship with the sinus, and the fangs of these teeth project into the floor of the sinus.

The sphenoid sinus has a close relationship with the cavernous sinus, optic chiasma, optic nerves and the hypophysis. For this reason sphenoid sinusitis was thought to be a cause of optic neuritis, and sphenoid surgery was immediately performed regardless of whether the patient had any findings of sphenoiditis. A few of the patients did get well.

The nasal conditions which may cause headache are those that produce pressure. Marked deviation of the nasal septum and polyps in the nose large enough to cause pressure may cause unilateral headache. One should never expect a deviation of the septum to the left side or polyp in the left side of the nose to cause headache on the right side.

Sinusitis itself may cause headache. If the infection is an overwhelming one and the ostia, the normal drainage tract of the sinus is blocked off, then pressure can be built up within the sinus causing headache. Pressure can develop within the sinus from a cyst or from a tumor

or actual invasion of the sinus wall, or destruction of bone by a carcinoma, such as a Schmincke's tumor. The headache must be on the same side as is the disease.

There are other factors beside the nose and the sinuses which may cause headache that are related to the ear, nose and throat. There is the so-called sphenopalatine ganglia neuralgia, which has a wide distribution. For many years it was the practice of otolaryngologists to remove or to exenterate the sphenopalatine ganglion when patients complained of unilateral head pain. More recently, the ganglion has been injected. It is not known why the procedure helped a few patients.

Glossopharyngeal neuralgia is a pain which usually starts at the root of the tongue and is referred to the ear. A large styloid process may cause similar symptoms. There is a syndrome which occurs when the geniculate ganglion is disturbed and bleb-like formations occur on the drum and the canal wall with an associated facial paralysis. Usually there is a severe headache on the same side.

It is very seldom that the otolaryngologist finds a cause for headache in the average patient. Nasal disease and sinus disease are seldom the cause of chronic headache in my experience.

MODERATOR MERRITT: The second speaker on the program is Dr. Frank Payne, Assistant in Ophthalmology, College of Physicians and Surgeons, Columbia University; Assistant Attending Ophthalmologist, Institute of Ophthalmology, the Presbyterian Hospital.

DR. FRANK PAYNE: It is true that the ocular examination often reveals signs such as papilledema, visual field defects and muscle palsies which are of great importance in diagnosing headaches due to intracranial diseases. I shall limit my remarks, however, to the headache and head pain due to diseases or abnormalities of the ocular apparatus.

Regarding the incidence, it is difficult to state how often headaches are due to the visual apparatus. Patients usually go directly to the oculist without first consulting the general physician, because the public knows that the eyes may be the cause of headache. Also, it is relatively easy for the patient to undergo an eye examination.

Two questions confront the general physician: 1) Which patients should be sent to the oculist? and 2) What are the relations between ocular headaches and the patient's systemic condition? Certain general principles may help in answering these questions.

The most common causes of severe ocular headache are acute congestive glaucoma and acute iritis. Even today these diseases, if accompanied by nausea and vomiting, are occasionally mis-diagnosed as acute disease of the gallbladder and are referred to the general surgeon before the eye is recognized as the cause of the symptoms.

The less severe and chronic, recurring headaches are due usually to refractive errors, anomalies of the accommodation and convergence mechanisms, and muscle imbalances. We do not expect the non-oculist to recognize these conditions if of low degree, but it is interesting and very significant that severe symptoms are commonly due to *small* errors of refraction, and *small* degrees of muscle imbalance; and they are the most difficult to detect. Apparently, an individual does not make the continued effort to overcome the large refractive errors and muscle imbalances and thus does not develop eye fatigue or a feeling of strain; he will, however, make the effort repeatedly to overcome the small errors, and headache ensues.

To suspect the eyes as the cause of a headache, certain things are worth knowing. The level of visual acuity does not indicate whether or not the headache is of ocular origin. A good acuity may be attained in the presence of a high degree of astigmatism; or, a patient may have a very low acuity and yet not have a headache. Thus, the presence of good vision does not exclude the eyes as the cause of the headache.

These ocular abnormalities are causing an increasing incidence of headaches because of the nature of our modern civilization. When we reflect that most of our civilization has evolved and has been designed upon the unconscious assumption that every adult has 20/20 visual acuity, and the ability to read fine print, we realize that the ocular demands are stringent. Our world is mostly within two feet of our face in our occupations and pleasures. Publications of all kinds are employing fine type which frequently is too small to be seen by older individuals. Since people are living longer lives it will become increasingly important for publishers to use print-size compatible with the visual acuity of older individuals. Television, colored movies and other entertainments add more demands upon our eyes. Whether or not an ocular anomaly is the cause of a patient's headaches can be determined first by finding that anomaly, and then correcting it. Two individuals may have an equal error of refraction and yet one individual has headaches and the other does not.

The patient's general state of fatigue, his temperament, and his visual requirements influence the role of the ocular anomaly. Often a person develops symptoms at the end of a school year, and then becomes comfortable during the summer's vacation.

Patients convalescing from serious systemic illnesses often have headaches when reading to pass the time pleasantly. Ocular fatigue and headache necessitate glasses, at least temporarily. It is wise to minimize the time that a patient may devote to reading when recovering from a systemic illness. This is of particular importance in children.

Another factor in the use of the eyes has to do with the illumination. I see many patients whose ocular symptoms are attributable to the fluorescent type of lighting. Fluorescent light is being installed in schools, factories, institutions and public buildings. In my sincere opinion it is the most inferior, most obnoxious and ugliest type of light ever devised. Architects and interior decorators would improve our world tremendously if they would return to the use of the ordinary type of incandescent light.

In conclusion, I believe that it is wise to advise an examination of the eyes in most cases of headache. The ophthalmologist should not conclude that an ocular anomaly is necessarily the cause of the headaches, but must consider also the patient's habits, general physical condition, ocular requirements and environment, and cooperate with the general physician to determine the true cause of the headaches.

MODERATOR MERRITT: Our third speaker is Dr. Arnold P. Friedman, Associate Professor of Clinical Neurology, College of Physicians and Surgeons, Columbia University; Attending Physician, Neuropsychiatry, Montefiore Hospital.

DR. ARNOLD P. FRIEDMAN: *Introduction:* The two most common headaches that confront the physician are migraine and tension headache (muscle contraction headache). In recent years new data have been made available through experimental and clinical investigation which have given the physician a better understanding of the diagnosis and treatment of migraine and tension headache.

MIGRAINE

A. Cause: The underlying causes of migraine are unknown. Many theories have been advanced but the large variety of precipitating fac-

tors and diversity of symptoms make it most difficult to explain all cases of migraine on any single etiologic basis.

Endocrine disorders, vitamin deficiencies, ocular malfunctions, trauma to the head and neck, allergy, hepatic disorders and cerebral anoxia have been proposed as the etiologic mechanisms in migraine headaches, but evidence to support these theories is lacking. A more tenable hypothesis is the inability of such patients to react adequately to stress for two reasons:

First, there is a hereditary factor; the mechanism through which this hereditary factor operates has not been clearly elucidated, but is possibly related to an imbalance of the autonomic nervous system. Secondly, they have an insecure personality pattern which interferes with their adjustment to their environment. This sets a pattern of function which the limited physiologic capacity of the individual is unable to handle, resulting in cranial vascular changes which produce the headache.

B. Mechanism: 1. Physiologic—Well substantiated observations from various sources have demonstrated that in an attack of migraine the following physiologic changes occur. An initial vasoconstriction of certain intracranial branches of the internal carotid artery produces visual and possibly other pre-headache phenomena prior to the onset of the headache. This prodromal period is followed by dilatation and distention of cranial arteries, primarily in the distribution of the external carotid artery. Stimulation of pain-sensitive nerves in and around the dilated vessels by the increased amplitude of pulsation is the presumed cause of the headache. Persistent dilatation results in a rigid, pipe-like state of the vessels. The pain at this stage is a steady ache replacing the earlier throbbing, pulsating type. During or following this, there is contraction of the neck muscles and “muscle-contraction pain” develops. This spasm of the muscles is a reaction to the initial pain and may outlast it. The initial phase of the headache is due to stimulation of pain endings which lie in or near the walls of the intracranial arteries, whereas the latter or “muscle-pain” is probably the result of either direct stimulation of nerve endings or ischemia of the muscles. Although in migraine the immediate cause of pain is associated with dilation of cranial arteries, it is evident that dilatation of blood vessels alone is not sufficient to produce headache. Other dynamic or chemical factors as yet not clearly delineated must contribute.

2. Psychologic—The importance of psychologic factors in the migraine attack is great. Personality studies have indicated that many patients with migraine are meticulous, neat in appearance, rigid in their thinking, and excessively aggressive toward their environment. It is often noted that in their earlier life they have had considerable insecurity with resulting tensions which are manifested in inflexibility, overconscientiousness, meticulousness, perfectionism and resentment.

In a recent study it was observed that psychodynamic mechanisms play an important role, even in migraine of young children. The types of neurotic symptoms observed in children were varied and dramatic. They included temper tantrums, phobias, frequent nightmares, hair-pulling, enuresis, obvious anxiety, hyperactivity, lethargy, thumbsucking, nailbiting and feeding difficulties.

In patients of all ages a variety of psychogenic factors have been related to the headache. Attempts have been made to define specific personality patterns. In our experience the personality make-up in these people is extremely variable and may be influenced by a variety of emotional factors. Most of these are unconscious and include hostility, identification with a family figure, a wish to remain in a position of dependency, or a desire to gain love, affection or attention. The most frequent conflicts are apparently concerned with hostile impulses associated with feelings of guilt.

There is little evidence of specificity of the precipitating psychodynamic factors. Not all patients with migraine are compulsive, perfectionistic or rigid. A recent objective psychologic evaluation of our patients confirms this point of view. Repressed hostility is an extremely common factor among many persons who do not have migraine but may have hypertension, ulcers or just a small bank account. Nevertheless, an understanding of the underlying psychologic factors plays an important part in the management of migraine, for in the ability of the patient to handle emotional tension lies the most satisfactory means of preventing the attacks in the majority of cases.

C. Characteristics: Migraine may be defined as that form of headache which is characteristically paroxysmal, periodic, unilateral, and throbbing. The headache occurs against a background of relative well-being, is often preceded by visual or psychologic disturbances, and is usually associated with vomiting and irritability. Frequently there is a history of similar headaches in the parents or other members of the

family.

The clinical picture of migraine indicates widespread bodily disturbances. Although attention is usually first called to the headache, there are numerous other symptoms to be considered as part of the entire picture. Among these are visual disturbances, including photophobia, autonomic disturbances such as pallor and sweating, edema of lids and extremities and difficulty with memory and concentration.

D. Objective Evaluation of Patients: General physical and neurological examinations are usually non-contributory. Routine laboratory studies such as blood counts, urinalysis, and skull roentgenograms are equally unrevealing. Ophthalmologic evaluation, including visual field studies, only rarely revealed any disorders. In our patients no striking variation from the normal electroencephalogram was noted in most cases, although Dow and Whitty indicate that a large number of abnormal records without specific patterns occurred in their migraine patients. Engel and his associates were able to demonstrate electroencephalographic changes accompanying the prodromal visual scotomas.

We were unable to find any relationship between fluid retention per se and the development of headache, nor were we able to relate migraine to a dysfunction of the pituitary adrenal system on the basis of eosinopenic response. Measurement of the volume changes occurring in the external carotid circulation in patients with migraine by use of the glycerine-pellotte and photo-electric cell technique gave us methods of recording objectively the changes in the external cranial circulation prior to and during the headache. Our observations with these tests indicated that the increase of the pulse wave amplitude was related to the severity of the migraine attack and following the use of a vasoconstrictor (ergotamine) there was a diminution of the pulse volume with a concomitant reduction in headache.

TENSION (MUSCLE CONTRACTION) HEADACHE

A. Cause: The actual cause of tension headaches is unknown. However, there is good evidence that they are related to psychologic disturbances. In a majority of our cases the fundamental psychic factors were largely unconscious, although most patients were aware of their anxiety. Environmental demands of an economic, social, physical or intellectual nature beyond the capacity of the patient's personality may also produce somatic response of which headache is the major

symptom. As in migraine the most frequently observed conflicts in cases of tension headache were those over hostile impulses. These patients demonstrated aggression, hostility and intense resentment against members of their family or persons who represent family figures. However, there is little evidence of specificity of the precipitating psychodynamic factors and as noted in migraine they may include identification, wish to remain in position of dependence, means of gaining attention, and so on. Although these findings help us to understand the mechanism of certain headaches, they do not elucidate the cause. Why the same psychogenic factors previously discussed as initiating headaches through physiologic mechanisms may also produce them on a purely psychologic level, such as in conversion mechanisms, is unknown. Furthermore, the same type of psychologic conflict may produce a variety of symptoms, such as headache, ulcers and hypertension, or may be present in an individual who is symptom-free.

Our present knowledge indicates that in tension headache, emotional conflicts which the patient has suppressed or repressed because of their unpleasant nature may produce a discharge, either skeletal or autonomic, with resultant changes in the muscles and blood vessels of the head and neck eventually productive of pain. The end organs for pain near these muscular and vascular changes are stimulated and create nerve impulses that pass along the afferent system to the central nervous system, where they are translated as painful sensations.

Muscular headaches differ from tension headaches in that the former occur with local muscle or nerve root injury, and secondarily, from noxious stimulation elsewhere in the head. The clinical features of the conditions causing the muscle spasm, for example, cervical disk, osteoarthritis, cord tumor, ocular inflammation, and so on, usually serve to differentiate these types of muscular headache from spasm associated with emotional tension.

B. Mechanism: In tension headaches muscular or vascular mechanisms may act independently or concomitantly. With muscle tension, sustained contraction of the skeletal muscles of the head and neck causes pain or dysesthesia in the neck and scalp. Associated with these muscular spasms may be ischemia which could be a contributory or primary factor in the induction of pain. It has been also hypothesized that excessive concentration of potassium in muscle from ischemia or sustained contraction stimulates the chemoreceptors in the tissues. An-

CHARACTERISTICS OF MIGRAINE AND TENSION HEADACHES (2000 CASES)		PERCENT	
		Migraine	Tension
FAMILY HISTORY OF HEADACHE		65	40
AGE OF ONSET	less than 20 yrs.	55	30
	over 20 yrs.	45	70
PRODROMA		60	10
FREQUENCY	constant or daily	3	50
	less than 1 week	60	15
DURATION	constant or daily	0	20
	1 to 3 days	35	10
TYPE	throbbing	80	30
LOCATION	unilateral	80	10
	bilateral	20	90
ASSOCIATED SYMPTOMS			
	vomiting	50	10

Fig. 1.—Migraine and Tension Headaches did not differ significantly in respect to the following: incidence, sex, precipitating causes, occurrence (time of day or season), mood change, onset and termination (abrupt or gradual).

other factor responsible for the head pain may be a central spread of the excitatory effect of noxious stimulation of the soft tissues of the neck. This spread of pain is carried by the upper cervical nerves and may produce painful sensations in the forehead and face.

Under stress a discharge may occur in the autonomic nervous system with resultant changes in the cranial blood vessels. The end organs for pain surrounding these blood vessels are stimulated and create nerve impulses which pass along the afferent pathways to the central nervous system.

C. Characteristics: Tension headache is that type of headache occurring in relation to constant or periodic emotional conflicts concerning which patients are partially aware. Tension headaches have no prodroma, are usually bilateral, occipital or frontal, and may be accompanied by a variety of associated signs, including anxiety, nausea and vomiting. Frequency and duration are variable.

D. Comparison of Migraine and Tension Headache: In comparing migraine and tension headache certain clinical features appear to be characteristic of each, although there are wide variations in each group and the same individual may have both types of headaches (Figure 1).

E. Differential Diagnosis: It is important to differentiate migraine and occasionally tension headache (muscle contraction headache) from other clinical conditions in which headaches are associated.

Tumors, particularly angiomas which most often appear in the parietal-occipital region, may lead to attacks of headache, nausea and vomiting, sometimes associated with visual hallucinations. Often convulsive seizures are part of this picture, and sooner or later intracranial pressure may develop as the tumor exerts pressure or encroaches upon neighboring parts of the brain. Sometimes in the case of angioma a cranial bruit may be heard. Migraine may simulate or be simulated by an aneurysm of the internal carotid artery which presses on a nerve. Persistent field defect and reduction of visual acuity or extraocular palsies may be associated with other focal signs in the case of the aneurysm.

Headache associated with temporal arteritis may occasionally be confused with migraine.

Transitory attacks of paresthesia, weakness and aphasia may occur in migraine without headache. Differentiation of this from disseminated sclerosis or cerebral vascular lesions may at times be difficult. Of course, headaches occurring alone must be distinguished from pain in the head due to other causes. In such headaches we must distinguish the various neuralgias, neuritides, diseases of the bones of the cranium, subdural hemorrhage, meningeal irritation, fibromyositis, etc.

Although rare, migraine is occasionally confused with glaucoma. The importance of measuring the intraocular pressure in headache patients cannot be over-emphasized.

TREATMENT

Treatments of migraine based upon a multiplicity of etiologies are remarkable for their consistent therapeutic success. In our experience the following is the most effective treatment for migraine:

1. Symptomatic—Symptomatic treatment of migraine is most effective by use of oral or rectal ergotamine tartrate and caffeine (Cafergot). Rectal use of Cafergot has proved empirically to be most efficacious, especially when oral medication cannot be retained. It is likely that rectal medication has the advantage of being absorbed more directly into the systemic circulation, without having to penetrate the hepatic and gastric barriers. Hence it is postulated that its action is quicker

with less side effects than by oral administration. This requires further investigation.

In some patients ergotamine tartrate is poorly tolerated and the use of dihydroergotamine methanesulfonate (D.H.E.-45) is indicated. Dihydrogenation of ergot reduces the vasoconstrictive action. In order to produce an equivalent therapeutic effect dihydroergotamine must be given in doses twice as great as ergotamine tartrate. It can only be administered parenterally, and like ergotamine, must be given as early as possible in the attack. Efforts to abort the attack in the prodromal phase by the use of vasodilators such as amyl nitrite, nicotinic acid, oxygen and B-methylacetylcholine (Mecholyl), etc., have been uniformly unsuccessful.

Analgesics and sedatives are sometimes indicated if the headache has been present long enough for edema to take place and the vessels to become firm and tortuous.

2. Prophylactic—It is well known that suitable psychotherapy may be of great value in reducing the intensity and number of migraine attacks in most patients and may even result in their disappearance for a considerable time.

Adequate relaxation, improvement in sleep and correction of any physiologic abnormalities will be of help in reducing the frequency of the attacks. In a few cases there may be evidence that the precipitating mechanism is allergic, endocrine, or "metabolic". It has been my experience that these factors are too isolated to be of real value in therapy. However, when an offending allergen is responsible it should be removed or the patient desensitized. If the onset of migraine is associated with the menstrual period the use of progesterone or testosterone, or both, may be helpful. Occasionally, a diet that restricts the daily intake of salt in addition to a diuretic seems to be beneficial.

The use of such drugs as histamine, nicotinic acid, etc., in my experience, is of no value. Sedatives to reduce emotional tension are limited in their usefulness. More recently we have used reserpine and Thorazine as interim treatment with migraine patients. Our results have not been encouraging.

Periodic and often paroxysmal nature of migraine and other types of vascular headache brought forth the idea that treatment with anti-convulsants might be of value in these disorders. Of the anticonvulsant drugs, two which we have had experience with are Dilantin and Mesan-

toin. Both are hydantoinates. These are analogous to the barbiturates, being derivatives of glycolyl urea as compared with malonyl urea. A small number of migraine sufferers respond well to Mesantoin, or Dilantin prophylactically. This limited group consists of patients who have an aura of aphasia, paresthesias, hemiplegias prior to the headache and an abnormal electroencephalogram. In certain other cases where there is a family history of migraine and epilepsy and an abnormal electroencephalogram, the use of certain anticonvulsant drugs is of value in controlling the headache. Dilantin and Mesantoin are administered orally.

TENSION HEADACHE

1. Symptomatic treatment—Symptomatic relief from tension headache is secured by drug therapy, which as in psychotherapy, must have flexibility for its keynote. The treatment of tension headache is non-specific, but the purpose of therapy is to relieve tension and raise the pain threshold. This is best accomplished by use of an analgesic-sedative combination. Such a combination should react with the minimal side effects allowing the patient to maintain his environmental relations and not be a problem because of addiction potential or development of tolerance.

2. Prophylactic treatment—Control of tension headaches can best be accomplished by use of psychotherapy, for this is the only method in which the patient's emotional conflicts can be resolved.

The use of one per cent procaine directly into the tender areas of the neck may eliminate the headache for several hours or a few days. Its long term use in tension headache is not consistent with the underlying mechanism causing the headache.

The use of sedatives such as barbitals and analgesics is only of temporary effect. Furthermore, their side effects curtail prolonged usage.

More recently we have used reserpine and chlorpromazine with patients with tension headache. Preliminary study indicates that reserpine is moderately effective.

In concluding, I would like to emphasize that the treatment and diagnosis of chronic headache do not belong to any one discipline or group in medicine. Rational therapy starts with the prerequisite that the physician be able to differentiate the type of headache that he is dealing with. To make the differentiation the clinician should under-

stand what is known about the underlying physiologic and psychologic mechanisms associated with chronic headache. In addition, of course, a detailed history and thorough examination are essential.

MODERATOR MERRITT: Our last speaker is J. Lawrence Pool, Professor of Neurological Surgery, College of Physicians and Surgeons, Columbia University; Attending Neurological Surgeon and Director, Department of Neurological Surgery, Presbyterian Hospital.

DR. J. LAWRENCE POOL: The cases I wish to discuss today are traumatic headaches and headaches of miscellaneous type and origin, including those due to cancer, aneurysms and brain tumors.

Post-traumatic headaches are frequent, as you well know. In my experience they are most frequent in those patients who are concerned with Workman's Compensation or litigation, or in patients with a neurotic or psychoneurotic tendency. Over the years, I have seen many patients with equally or more severe head injuries than many of the litigious and neurotic patients, who nevertheless have not had any headaches at all. As I have studied that aspect of the problem it seems to me that a very distinct feature in many cases of post-traumatic headaches is a psychological or psychiatric factor. One also sees patients with headaches after head injury due to local scalp injury, if there has been a laceration resulting in a painful neuroma. That is rare but does happen. The treatment may require local procaine injection or excision of the painful area. Subdural hematomas may give headaches of unilateral origin or location. One finds a few patients who suffer chronic recurrent headaches after head injury which are relieved by pneumoencephalogram, probably because drainage of cerebrospinal fluid alters the intracranial fluid pressure and relieves tension of adhesions upon the dural structures lining the skull. This measure is not effective in most cases, however. With these head injuries and post-traumatic headaches it is important to examine the neck for possible cervical injury. As Dr. Friedman pointed out, secondary muscle spasm may be the cause of severe headaches in the occipital region. This may be initiated by a subluxation of one vertebra on one side only, and x-rays of the neck should therefore be obtained when no other cause for these headaches can be found.

Turning to miscellaneous conditions that the neurosurgeon encounters, "temporal arteritis" was formerly seen a good deal. I think it fits

into the group that Dr. Friedman described. However, we do not seem to see such cases or treat them by surgery any more.

Tic douloureux or trigeminal neuralgia is so familiar to you that treatment by alcohol block or differential root section need not be discussed. Glossopharyngeal tic or 9th nerve pain at the root of the tongue and base of the throat is an extremely painful, paroxysmal condition that can readily be relieved only by section of the 9th nerve intracranially through a small opening. This is a simple and safe procedure. The newest treatment for trigeminal neuralgia is simple compression of the posterior root without cutting any of the pain fibers. This has proved extremely effective, so that little or no sensory loss occurs and pain is relieved. Carcinoma pain due to invasion of the base of the skull, as Dr. Baker has mentioned, may be relieved in some cases by sectioning of a number of cranial nerves on the involved side. This is a major procedure that involves sectioning the posterior root of the 5th nerve because the trigeminal innervates the dura, also the 9th nerve and also the first three cervical sensory roots on that side. All of this can be done through a simple posterior or suboccipital incision. It does reduce the pain a great deal but I have never seen it reduce the pain of cancer in the base of the skull completely. One may therefore also have to consider a unilateral lobotomy in such patients and this, combined with nerve section, has given a number of my patients relief, although not complete relief, during the months of their survival.

Intracranial aneurysms may also give pain. They may give pain referred to the eye, which is often excruciating in severity. It is paroxysmal and as a rule, feels as if, as the patients say, a knife is being twisted around back of the eyeball. Indeed, there may be no other symptoms or signs of such an aneurysm. Carotid angiography should therefore be considered in such cases, a simple and effective diagnostic test. The treatment of aneurysms is ligation of the internal carotid in the neck and trapping the aneurysm when possible by placing a clip on the vessel inside of the head at the circle of Willis. This depends on the location of the aneurysm and its nature.

Turning finally to brain tumors, we find that headaches are commonly caused by increased intracranial pressure but the presence of a tumor without pressure may also cause headaches. I would like to stress this because many of us, when we see such patients and find no

papilledema, have said that there could be no brain tumor because there was no increased intracranial pressure. However, in a recent study of acoustic nerve tumors we have found no increased intracranial pressure accompanying this type of tumor. Headache is probably due to pressure upon the tentorium or dural membranes. Postural headache, of course, may be produced by any type of brain tumor, with increased pressure, including 8th nerve tumors and intraventricular tumors.

I might say in closing then, that the treatment of many of these headaches due to intracranial lesions of the sort mentioned, particularly brain tumors, is carried out now in a much safer manner than in former days when the mortality rate from neurosurgery was high. Today we expect good results and get them.

MODERATOR MERRITT: I think it would be of value to summarize the remarks made by the members of the panel. Dr. Baker and Dr. Payne are unusually modest representatives of their specialties because they both stated that although headache could be caused by disease of the structures in which they are particularly interested, disease of these structures was not an important factor in the majority of the patients with headaches who come to the practicing physician. This statement is also borne out by Dr. Friedman's discussion. All of the speakers have stressed the importance of the thorough study of the patient from the physical side and Dr. Friedman emphasized the need for a careful psychological study. Drs. Baker and Payne emphasized that, in the treatment of these patients, relief of symptoms is not to be expected from treatment of the eye or nose if there is no significant disease of these structures. The vast majority of patients who come to the physician complaining of headache fall into one of three groups: migraine, tension headaches, or traumatic headache.

It is important to note that constant headaches are practically never a symptom of organic brain disease or organic disease elsewhere in the body. If a patient complains of headache that persists for weeks, months and years without let-up, it is probable that it is related to emotional tension.

The speakers have all stressed the importance of a thorough examination, including the eye, nose, throat, ear, the nervous system and the body in general, and that if any abnormalities are found in these examinations, they should be taken into consideration and properly treated.

In general, the treatment of the patient with chronic headaches is divided into two parts: 1) symptomatic, that is treatment of the headache when it occurs; and 2) prophylactic, the prevention of subsequent attacks.

In patients with tension headaches and practically all other types, except migraine, analgesics and sedative drugs give the best results.

Ergot preparations are the drugs of choice during an attack of migraine headache. In prophylaxis, in the prevention of recurrences, drugs are of little value. Measures directed toward the relief of emotional tension and psychotherapy to assist in the solution of every-day problems are of considerable importance. Dr. Friedman has correctly stated that this type of therapy is in the province of all doctors. If the physician gives the patient sympathetic understanding and allows the patient to do some of the talking, good results can be obtained.

We have a number of questions from the audience. Dr. Pool, is vitamin B₁₂ of value in the treatment of trigeminal neuralgia?

DR. POOL: I don't think it is. It is worth a trial but our experiences with the use of vitamin B₁₂ have not been encouraging.

MODERATOR MERRITT: *There are a number of questions, all dealing with the same subject, that is, what is the preparation that is used in the form of rectal suppositories in the treatment of migraine? Dr. Friedman, can you tell us what the suppository contains and how it is used?*

DR. FRIEDMAN: Cafergot suppository—the average dose is one suppository (ergotamine tartrate 2 mg. and caffeine 100 mg.) every half hour, using a total of three suppositories if necessary. We have also found that the use of the above-mentioned dosage with 0.25 mg. Bellafoline is effective in many cases where abdominal spasm and nausea are complained of.

MODERATOR MERRITT: *This is for Dr. Pool. What are your views with regard to the treatment of postoperative headache and post-spinal puncture headache?*

DR. POOL: Post-spinal headache should be treated by keeping the head down and feet elevated for 48 hours. As to the postoperative headache, most people after craniotomy, even of large size, do not complain of headaches. They may complain of them at times when

worried, tense, fatigued. You would think they would have a lot of headaches. As a rule they don't have any.

MODERATOR MERRITT: *This question is for Dr. Baker: Has it been proven that there exists a "vacuum type headache", and what is the treatment?*

DR. BAKER: There probably is a vacuum type of headache. It is one type of headache we frequently see in individuals who are exposed to rapid changes in atmospheric pressure, as in airplane flight or deep sea diving. We know that such patients will often experience a severe type of headache. The headache brought on by the pressure changes may be severe and associated with actual hemorrhage within the sinus itself. There does not seem to be any other explanation for this particular type of headache.

MODERATOR MERRITT: *Treatment?*

DR. BAKER: The treatment for it, of course, is to try to get the nose and sinuses better ventilated by the use of a vasoconstrictor.

MODERATOR MERRITT: *Dr. Friedman, is there such an entity as histamine cephalalgia? Is desensitization with histamine useful?*

DR. FRIEDMAN: A clinical syndrome consisting of periodic unilateral headaches related to the periorbital region and associated with injection and watering of the eyes and stuffiness of the nose has been described by Bing, Horton, Harris and Gardner by various names. Horton described this headache as being caused by histamine and relieved by treatment with this same drug. It is our belief that this type of cyclic cluster headache is a variant of migraine and its relationship to histamine is questionable. These headaches are usually relieved symptomatically by use of a vasoconstrictor. In a few cases, histamine, cortisone and similar types of medicaments have been of value prophylactically. Due to the fact that the duration of these headaches is usually from four to eight weeks, it is difficult to evaluate prophylactic treatment.

MODERATOR MERRITT: *We have two more questions on therapy for Dr. Friedman. What dosage of reserpine have you found useful? Is there any delayed response? Also can you comment on the use of mephenesin compounds in tension headaches?*

DR. FRIEDMAN: The average daily maintenance dose is 0.75 mg. orally. In many cases, however, patients are effectively stabilized at 0.5 mg. There seems to be no appreciable difference in the end results whether the patient is loaded in the beginning of therapy or saturation achieved slowly on a constant dose. Maximal effective action in most cases is obtained after 7 to 10 days on therapy. The drug is well tolerated and only mild side effects are noted. Sleepiness and dryness of the nasal mucosa, nausea and diarrhea are complained of in a small number of patients after maintenance is established. No evidence of addiction is noted. Since the action of reserpine taken orally is cumulative and slow, it is generally used as a preventive or prophylactic medication and not for symptomatic treatment. We have not found Tolersol to be effective as interim treatment for tension headaches.

MODERATOR MERRITT: *This question is not addressed to any of the panel, but it might go to Dr. Baker: What is the cause of early morning headaches, not in a hypertensive person but in a person who likes to sleep late?*

DR. BAKER: I will take a chance in trying to answer it. Often in the morning when a person wakes up the nose is apt to be plugged up and filled with a lot of secretion and such a condition persists until the patient starts moving around. This is particularly true if he happens to sleep in a poorly ventilated room.

MODERATOR MERRITT: *We have two final questions. I think they go to Dr. Friedman: What is the best therapy for headache and dizziness due to cerebral thrombosis and hypertensive arteriosclerosis?*

DR. FRIEDMAN: In the treatment of cerebral thrombosis we are limited to the use of the vasodilators, such as nicotinic acid and papaverine. Analgesics such as salicylates and codeine may be used for symptomatic treatment.

In the treatment of hypertensive headaches, management of the general state of hypertension is always indicated, aiming at a decrease in the blood pressure and protection of the patient against vascular insults rather than merely elimination of the headache. The available measures for lowering the pressure include psychotherapy, rest, sodium restriction, and specific anti-hypertensive drugs. Antipressor drugs used for the reduction of blood pressure include the methonium salts, diaben-

mine hydrochloride, Hydralazine, hydrogenated ergot alkaloids and Rauwolfia serpentina. Recently we have used Rauwolfia serpentina in a group of over thirty patients with hypertensive headache. This drug acts as a hypotensive agent as well as a sedative. Although not a potent hypotensive agent it has proved to be effective in reducing the severity and frequency of the headache as well as a moderate lowering of the blood pressure. We are encouraged by our results with this drug but more time is needed to prove its efficaciousness.

MODERATOR MERRITT: *We have one last question. It is posed to Dr. Friedman: "I have a headache preceding a rain. Is there any prophylaxis?" I think probably this patient should go to some country where they don't have rain. Dr. Friedman, would you give a more intelligent answer?*

DR. FRIEDMAN: I believe that Dr. Merritt's suggestion is the most effective therapy. Patients complaining of cephalalgia with temperature, humidity and atmospheric pressure changes are believed by some to suffer from a physical type of allergy. The pain is usually muscular in origin and is said to be similar to that produced experimentally by exercising a muscle which is in an ischemic condition. It is our belief that this represents another form of stress headache. The treatment is non-specific and may include the use of vasodilators, physical therapeutic measures and analgesics.

MODERATOR MERRITT: On behalf of the Academy of Medicine, I wish to thank the members of the Panel for their contributions to this afternoon's program.